

The influence of physical fitness and exercise upon cognitive functioning: A meta-analysis

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Abstract:

Nearly 200 studies have examined the impact that either acute or long-term exercise has upon cognition. Subsets of these studies have been reviewed using the traditional narrative method, and the common conclusion has been that the results are mixed. Therefore, a more comprehensive review is needed that includes all available studies and that provides a more objective and reproducible review process. Thus, a meta-analytic review was conducted that included all relevant studies with sufficient information for the calculation of effect size ($N = 134$). The overall effect size was 0.25, suggesting that exercise has a small positive effect on cognition. Examination of the moderator variables indicated that characteristics related to the exercise paradigm, the participants, the cognitive tests, and the quality of the study influence effect size. However, the most important finding was that as experimental rigor decreased, effect size increased. Therefore, more studies need to be conducted that emphasize experimental rigor.

Keywords: mental ability | intelligence | physical activity

Article:

Historically, the relationship between the mind and body has been studied from either a dualistic or a holistic perspective. Early Greek and Hebrew philosophers adopted a dualistic perspective and viewed the mind and the body as two distinct entities. However, since then, most philosophers and researchers have taken a holistic perspective, considering the mind and the body as two interrelated, inseparable entities. In recent years, controversy has shifted from the question of whether there is a link between the mind and body to the question of what the precise causal relationship is between these two components. In particular, one line of research has focused upon the idea that there is a causal link between the body and the mind such that exercising the body has a beneficial effect on the performance capabilities of the brain.

Piaget (1936) was one of the first to suggest that motor development is an important determinant of intellectual development in children. Other early theorists have also adopted this holistic approach and have come to similar conclusions. For example, the perceptual-motor concepts of Kephart (1960) suggest that children who have learning disabilities may actually be suffering from perceptual-motor problems. Additionally, Gestalt psychologists have influenced the entire

field of education through their emphasis on the idea that "an individual functions as a whole within the environment and thus must be treated accordingly" (Kirkendall, 1986).

Although theories have been proposed that suggest that the body can influence the mind, until recently, actual mechanisms that could support this causal link were not known. Contemporary researchers examining the influence of exercise on cognitive functioning have taken advantage of advances in the field of neuropsychology and have used animal research to discover the mechanisms that could explain this link. One such mechanism is cerebral blood flow. Research on humans that has used modern methods (i.e., Xenon clearance techniques) and moderate- to-high intensities of exercise has shown large increases in cerebral blood flow as a function of exercise (Herholtz et al., 1987; Jørgensen, Perko, Hanel, Schroeder, & Seeber, 1992; Jørgensen, Perko, & Secher, 1992; Thomas, Schroeder, Secher, & Mitchell, 1989). It has been suggested that these increases in cerebral blood flow then benefit the cognitive functioning of the organism because they result in an increased supply of necessary nutrients to the brain (e.g., glucose, oxygen) (Chodzko-Zajko, 1991; Jacobs, Winter, Alvis, & Small, 1969; Madden, Blumenthal, Allen, & Emery, 1989; Speith, 1965).

Another mechanism that could explain a causal link between the body and the mind is the influence of exercise upon brain neurotransmitters. Several authors have shown that neurotransmitter levels change as a function of an acute bout of exercise. Investigators have found increases in norepinephrine or its precursors (Ebert, Post, & Goodwin, 1972; Gordon, Spector, Sjoerdsma, & Udenfriend, 1966; Mitchell, Flynn, Goldfarb, Ben-Ezra, & Copmann, 1990), in serotonin (Barchas & Freedman, 1963), and in endorphins (Bortz et al., 1981) after an acute bout of exercise. Researchers examining the influence of chronic exercise upon neurotransmitters have also found more long-term increases. As a function of chronic exercise programs, Poehlman and Danforth (1991) and Poehlman, Gardner, and Goran (1992) found increases in norepinephrine in humans, and Brown et al. (1979), Brown and Van Huss (1973), and Radosevich et al. (1989) found increases in norepinephrine in animals. These findings are important in terms of cognition because research with mice has shown that high levels of norepinephrine are associated with better memory (Zometzer, 1985) and because the norepinephrine and dopamine synapses appear to play important roles in memory (Kalat, 1992).

Another physiological explanation for how exercise may affect the mind is that exercise may result in permanent structural changes in the brain. In two related studies (Black, Isaacs, Anderson, Alcantara, & Greenough, 1990; Isaacs, Anderson, Alcantara, Black, & Greenough, 1992), rats that were exposed to exercise had an increase in the density of the vasculature in the cerebellar cortex and had shorter vascular diffusion distances as compared to nonexercising controls.

Although the neuropsychological research examining these mechanisms in humans is limited due to the cost and invasiveness of the techniques, the initial results indicate that (a) exercise may result in changes either in the brain itself or in the brain environment, and (b) these changes may have a positive influence on the performance capabilities of the brain (Dustman, Emmerson, & Shearer, 1994). Additionally, a great deal of research has used a behavioral approach to examine the influence of exercise or fitness level upon cognitive functioning. In fact, nearly 200 behavioral studies have been conducted to examine this relationship. The results of these studies

have been quite varied and, for this reason, these studies have been reviewed using traditional narrative review techniques by 11 authors (or groups of authors) in an attempt to come to an overall conclusion. In attempting to understand the relationship between exercise and cognitive functioning, it is worth while to discuss the existing reviews of this literature.

Several authors have reviewed the results of studies that have looked at correlations between physical fitness and mental achievement. In an early review, Clarke (1958) summarized the results of seven studies and concluded that all of the results were in a positive direction. In contrast, Harris (1973) reviewed the literature on motor performance and academic achievement and concluded that the relationship between physical activity and academic achievement had not been established. Later, Kirkendall (1986) divided the literature into two different groups of studies. From the studies that compared intelligence and academic achievement between athletes and nonathletes, Kirkendall concluded that there was a "modestly positive" relationship between athletic success and academic success. However, from the studies that attempted to correlate motor and intellectual performance, Kirkendall concluded that physical growth measures are poor predictors of intellectual performance. That same year, Thomas and Thomas (1986) reviewed studies examining perceptual-motor development, physical education, and athletics and their relationship to academic performance. Thomas and Thomas concluded that none of these activities enhanced academic performance. Finally, Anthony (1991) reviewed the psychological aspects of exercise and concluded that a high correlation exists between exercise participation and intellectual and memory capabilities.

Two other authors reviewed studies in this same area and chose to qualify their conclusions based upon the nature of the cognitive task or the nature of the exercise. Weingarten (1973) qualified his conclusions based upon the complexity of the cognitive task. He concluded that fitness has a positive impact on the performance of complex cognitive tasks, but that fitness has no impact on the performance of simple cognitive tasks. Subsequently, Gruber (1975) examined the relationship between academic achievement and physical fitness and qualified his conclusions based upon the nature of the exercise. He concluded that exercise will only be beneficial to cognitive functioning if the exercise involves coordinated movements that require thought before execution and if these movements use the same structures and functions necessary for the cognitive performance.

Other authors have summarized the results of studies using acute exercise. Most of these authors also chose to qualify their conclusions based upon aspects of the exercise or of the cognitive measure. Gutin (1973) concluded that the complexity of the cognitive task and the duration of the exercise significantly affected the results. In particular, he suggested that with reaction time (RT) measures, the optimal level of exercise-induced arousal would be inversely related to the number of choices. Additionally, Gutin suggested that prior exercise of 45 s to 2 min (which brings the heart rate to 90-120 beats per minute) was beneficial to cognitive performance, whereas exercise that was 6 min in duration and that increased heart rate to 150 beats per minute was detrimental to performance. In 1975, Powell also reviewed the acute exercise literature and suggested that the intensity of the exercise significantly affected the results. He concluded that acute submaximal exercise can result in short-term improvements of cognitive functioning and that long-term training can serve as a buffer against the typical decreases in cognitive ability that

occur with age. However, Powell also suggested that acute exercise of maximal intensity may have a negative effect on cognitive ability.

In 1986, Tomporowski and Ellis conducted a more systematic and comprehensive narrative review of the literature on acute exercise and cognitive functioning. All of the available published studies ($n = 27$) were classified into groups based upon the duration and intensity of the exercise protocol. The authors concluded that studies that used exercise protocols of short duration and moderate intensity led to improved cognitive functioning.

There have been only two reviews dedicated to summarizing results from studies using chronic training protocols. Folkins and Sime (1981) examined 10 such studies and qualified their results based upon the age and health status of the participants used. They reported that studies of geriatric mental patients had shown positive effects of exercise upon cognition, but that studies of children or normal adults had not shown clear results. Hughes (1984) reported the results of three studies that had examined the effects of habitual aerobic exercise upon cognition. From such a limited number of studies, it is not surprising that Hughes could conclude only that there were mixed results.

These reviews provide good examples of the difficulty in reaching a definite conclusion regarding the relationship between exercise or fitness and cognition. In fact, the only consistencies among the 11 reviews has been the scarcity of research in the area and a general consensus that "the results are mixed." Fortunately, however, the statistical technique of meta-analysis can be used to more clearly summarize the results of numerous studies addressing a common research question. This technique is extremely beneficial because it allows for the simultaneous consideration of all available studies in a particular research area rather than being limited to a subsample of the available studies. Therefore, the results of this meta-analysis will provide information that cannot be found in single studies or in narrative reviews. Additionally, because the use of meta-analysis allows the results of numerous studies to be combined, this effectively combines the samples from the studies so that statistical power is increased. Finally, meta-analysis allows for the examination of moderator variables to determine if certain characteristics of the studies are associated with larger effect sizes.

Hypotheses

Researchers working in the area of exercise or physical fitness and cognitive functioning have identified several mechanisms that may mediate the relationship between exercise and cognition. Based upon these ideas, certain a priori hypotheses have been proposed in an attempt to either support or fail to support these potential mechanisms. Additionally, researchers have suggested that there may be limitations on the proposed positive relationship between exercise or physical fitness and cognitive functioning such that the positive relationship may only hold for certain tasks or in certain individuals. Therefore, a priori hypotheses have also been proposed to test these suggested limitations:

- Hypothesis 1: Based upon the mechanisms of increased neurotransmitters and changes in brain structure that have been documented with training studies, it is hypothesized that

studies using a chronic training program or making a cross-sectional comparison between fit and unfit individuals will show the largest effect sizes.

- Hypothesis 2: Based upon the suggestion that exercise may be most beneficial to those individuals who are mentally impaired (Folkins & Sime, 1981), it is hypothesized that studies using mentally disabled participants will show greater effect sizes than will studies using normal participants.
- Hypothesis 3: Evidence suggests that older individuals may be resource limited because they have less attentional flexibility and are not able to time-share as well as younger adults (Hawkins, Kramer, & Capaldi, 1992) or because older adults, as a group, have insufficient oxygen transport to the brain (Jacobs et al., 1969; Speith, 1965), have decreased neurotransmitter synthesis (McGeer & McGeer, 1980; Palmer & DeKosky, 1993), and have an overall decrease in cerebral metabolism (Marchal et al., 1992). Therefore, it is also hypothesized that studies using either elderly (61-90 years) or younger (less than 18 years) participants will show bigger effect sizes than will studies using middle-age (31-60 years) or college-age participants (18-30 years).

Several hypotheses specific to particular exercise paradigms could also be derived based upon conclusions made in the previously described reviews:

- Hypothesis 4: Based upon Gutin's (1973) review, it is hypothesized that acute studies in which high-intensity exercise was used will show greater effect sizes with measures of simple RT (RT to only one stimulus) than with measures of choice (RT to one stimulus when two or more are possible) or with discriminant RT (RT to a "go" stimulus when a "no-go" stimulus is possible), whereas the opposite will be true for studies in which low-intensity exercise was used.
- Hypothesis 5: Gutin (1973) also suggested the hypothesis that the duration of the prior acute exercise will show an inverted-CT relationship with effect size.
- Hypothesis 6: Based on the conclusions of Tomporowski and Ellis (1986), which suggests a slightly different hypothesis with regard to duration and intensity of the acute exercise, it is hypothesized that studies with acute exercise sessions of short duration (2-15 min) and moderate intensity will show the largest effect sizes compared to studies with exercise sessions of longer durations (> 15 min) or with difficult or light intensity.
- Hypothesis 7: In response to the theory that improvements in cognitive functioning as a function of exercise training are due to physiological changes in the brain, it is hypothesized that, among studies using chronic exercise paradigms, the longest training periods will show the greatest effect sizes.
- Hypothesis 8: With regard to cross-sectional studies, Weingarten's (1973) review suggested that differences as a function of fitness level will only occur with complex cognitive tasks (i.e., choice and discriminant RT vs. simple RT; IQ, reasoning, math, verbal vs. perception).
- Hypothesis 9: It is hypothesized that studies designed to examine the relationship between fitness and mental achievement (i.e., correlational or cross-sectional studies) will show relatively small (correlational: $0.10 < ES < 0.30$; cross-sectional: $0.20 < ES < 0.50$; Cohen, 1992) positive effect sizes (Clarke, 1958).

Method

Literature Search

Computer-aided searches were conducted using CD-ROMs developed for Psych-Lit, Educational Research in Completion (ERIC), Dissertations Abstracts, and Completed Research in Education. The key words used in these searches were *cognition*, *cognitive performance*, *mental*, *intellect*, *exercise*, *fitness*, *acute*, and *chronic*. Hand searches were also conducted to identify relevant articles cited in the reference lists of those articles found through computer searches. The literature search resulted in 176 studies that examined the influence of exercise on cognitive performance. Forty-two did not provide sufficient information for the calculation of effect size. That is, calculating an ES requires one of the following: (a) means, standard deviations, and sample sizes for the treatment and comparison groups; (b) the value of the statistical test (*r*, *t*, or *F*) and the sample sizes of the treatment and comparison groups; or (c) the significance level and the sample sizes of the treatment and comparison groups. The remaining 134 studies yielded 1,260 effect sizes for subsequent analyses.

Calculation of Effect Sizes

Several different methods have been proposed for the calculation of effect sizes. In this analysis, Hedges's (1981) formula was used for the calculation of effect sizes. This formula is: $ES = (M_E - M_C)/SD_P$, where

$$SD_P = \sqrt{\frac{(N_E - 1) \cdot SD_E^2 + (N_C - 1) \cdot SD_C^2}{N_E + N_C - 2}}$$

and where ES = effect size, M_E = mean of experimental group, M_C = mean of comparison group, SD_P = pooled standard deviation, N_E = number of participants in the experimental group, N_C = number of participants in the comparison group, SD_E = standard deviation of the experimental group, and SD_C = standard deviation of the comparison group.

Because studies with small samples sizes may have a biased effect size (Thomas & French, 1986), each effect size was then multiplied by a correction factor designed to yield an unbiased estimate of effect size (Hedges, 1981). This correction factor is: $c = 1 - [3/(4m - 9)]$, where $m = N_E + N_C - 2$.

Coding the Studies

To examine the moderating effects of particular variables upon the results, certain relevant variables were coded a priori. These variables were identified based upon the conclusions of the past reviews, the existing theories to explain the relationship between exercise and cognition, and the findings of past meta-analyses. For each categorical moderator variable, the study was coded with integer values so that those variables which might moderate the effects of exercise on cognitive ability could be examined. Continuous variables were also identified and coded numerically for subsequent analysis. Variables that related to characteristics of the participants,

the quality of the study, characteristics of the exercise, and characteristics of the cognitive measures were coded.

Participant Characteristics. The age of the participants was coded as unreported, 6-13 years, 14-17 years, 18-30 years, 31-45 years, 46-60 years, and 61-90 years. Gender was coded as male, female, or mixed. Additionally, mental fitness (normal, impaired) and physical fitness (trained, untrained) were coded to determine whether exercise has a differential effect on the cognitive performance of impaired individuals or on the performance of trained individuals.

Quality of the Study. Critics of meta-analysis have suggested that this technique is inherently flawed because it follows the principle of "garbage in, garbage out": If poorly designed studies are included in the analyses, the results of the meta-analysis will also be of poor quality (Kazdin & Wilson, 1978; Searles, 1985). Glass (1977), however, suggests that this can be controlled for by an a posteriori examination of the influence of poorly designed studies upon the resulting effect sizes. Therefore, studies were coded for variables that might affect their quality, including study design (between-subjects, within-subjects, mixed design, correlational), publication status (published, unpublished), sampling method (random stratified, random, intact group, volunteer, other), method of assigning participants (random stratified, random, intact group, single group, or matched), and number of threats to internal validity (Campbell & Stanley, 1963). In addition, studies were coded in terms of whether the comparison groups were equivalent with respect to relevant variables other than the treatment variable.

Exercise Characteristics. Because a variety of exercise protocols were followed in the studies obtained, variables were coded to describe the exercise protocol used. The primary difference between exercise protocols resulted from the use of chronic or acute exercise and from the use of quasi-experimental or true experimental designs. Therefore, effect sizes were coded as acute, chronic, mixed acute and chronic, or cross-sectional/correlational. For chronic exercise paradigms, evidence of improved fitness from training was coded as not reported, yes, or no. Additionally, the number of days per week that the participants trained and the number of weeks that they had trained were coded as continuous variables.

For all of the exercise paradigms, other variables examined were the number of sessions of exercise immediately prior to the cognitive test, the duration of the exercise session (in minutes), the intensity of the exercise, the type of activity performed, the exercise environment, the exercise group size, and the exercise leader. Number of sessions of exercise and duration of the exercise were coded as continuous variables. Intensity was coded as not reported, low intensity, moderate intensity, or high intensity. Activity was coded as not reported, aerobic, muscular resistance, isometrics or calisthenics, handgrip dynamometer, games with an aerobic component, games without an aerobic component, or step tests. Exercise environment was coded as laboratory, home, hospital, fitness center, class setting, organized team recreational, organized team competitive, and other. Exercise group size was coded as alone, 10 or less, 11-20, or more than 20. Exercise leader was coded as no leader, experimenter, fitness trainer, teacher, coach, or other.

Cognitive Measures. A wide variety of measures have been used to assess cognitive ability. Each particular cognitive test used was coded separately. Across the studies included, 106 different

cognitive tests were used, and these included simple reaction time, line matching tests, verbal comprehension tests, the Culture Fair Intelligence Test, the Stanford-Binet Intelligence Quotient, the Wechsler Memory Scale, the Sternberg Number Task, the Stroop test, and Raven's Progressive Matrices Test. Because so many different cognitive tests were used and because many of the tests were only used on minimal occasions, studies were further coded as to which type of cognitive ability was being assessed with the particular cognitive test. Thus, the cognitive tests in each study were classified as being in one of the following cognitive categories: memory, mathematical ability, verbal ability, reasoning, creativity, academic achievement, mental age, intelligence quotient, dual task paradigms, RT, motor skills, perception, or other.¹ Subsequent analyses with regards to the cognitive test were then done using cognitive category as the moderator variable of interest.

Analysis

All of the possible effect sizes that could be calculated were included in the overall average effect. However, this means that studies with multiple comparison groups or with multiple measurements of the dependent variables included many sets of dependent effect sizes. Since this violates the assumption of independent data points (Bangert-Drowns, 1986), using all of these data points is not desirable for statistical analyses. The only options to maintain this assumption are to combine all of the effect sizes from one study into an average effect size or to choose only one effect size as representative of each study. However, these two options result in the loss of information that can be gleaned from the studies because these options demand that levels of the moderator variables be either ignored or combined. Therefore, the most logical decision was to limit each study to one set of effect sizes from one comparison group. The order of preference for this set of effect sizes was determined by the comparison group as follows: (a) own pretest, (b) treatment control group, and (c) no-treatment control group. Following this, outliers were identified as those effect sizes that were greater than three standard deviations from the mean; these outliers were eliminated from subsequent analyses.

The next step in the analysis involved examining moderator variables to determine which aspects of a particular study might significantly influence the size of the effect. For the categorical moderator variables, one-way analyses of variance (ANOVAs) were conducted with corrected effect size as the dependent variable and with the various moderator variables as the independent variables. For the continuous moderator variables, correlational analyses were conducted. To control for Type I error, the Bonferroni correction was used so that the level of significance was adjusted based upon the number of simultaneous analyses. This resulted in the significance level being set at $p = .002$ because 19-22 analyses were done for each subset of the data. The technique of ANOVA was used because an examination of the distribution of the effect sizes showed a normal distribution which suggests that this technique is appropriate (Wolf, 1986).

For moderator variables that had a significant impact on the effect sizes, the Scheffe test was used for post hoc analyses to determine which levels of the variables were significantly different ($p < .05$) from each other and a z statistic was used to determine if the effect size was equal to zero ($p < .05$) (Cooper & Hedges, 1994). In interpreting the results, only those levels of the

¹ The names of the individual cognitive tests which were used and the cognitive categories into which they were encoded are available from the first author upon request.

variable which had five or more effect sizes were considered. This criteria for the number of effect sizes was based on the logic that results should not be interpreted when the effect sizes were all derived from the same study and that with five or more effects sizes it was likely that the effect sizes came from more than one study. However, it should be noted that in most instances the sample sizes in the levels of the ANOVA were not equivalent, and therefore, care should be taken in interpreting the results (Keppel, 1991). Moderator variables that did not significantly affect the results were not discussed. After determining which moderator variables had significant influences upon the effect sizes, these variables were included in a series of cross-tab analyses to examine the loading of the variables in the various categories so that potential third-order causation could be identified. Finally, the a priori hypotheses were examined specifically using the most appropriate statistical technique for each particular hypothesis.

Bangert-Drowns (1986) has suggested that separate analyses should be conducted when dependent measures represent different logical constructs. Therefore, the first moderator variable examined was exercise paradigm because of the concern with mixing the results of studies that had used different experimental designs. From then on, separate analyses were conducted for the studies within each exercise paradigm.

Results

The overall mean effect size for all of the studies was found to be 0.25 ($SD = 0.69$, $ES\ n = 1,260$, $p < .05$), which suggests that exercise improves cognitive functioning by 0.25 standard deviations and that this improvement is significantly different from zero. Following this initial computation, effect sizes from the best comparison group for each study were used for subsequent analyses. This resulted in the exclusion of 397 effect sizes, with 873 effect sizes remaining ($M = 0.30$, $SD = 0.78$, $p < .05$). Additionally, effect sizes that were greater than 3 standard deviations from the mean were labeled outliers and were omitted from subsequent analyses. This resulted in the omission of 21 effect sizes. Therefore, the adjusted overall mean was 0.29 ($SD = 0.63$), with 852 effect sizes included.

The nature of the exercise intervention used significantly influenced the effect sizes, $F(3, 848) = 12.43$, $p < .001$. There were only six effect sizes from studies using mixed designs that combined acute and chronic exercise ($M = 0.54$, $SD = 0.43$, $ES\ n = 6$, $p < .05$). An examination of the influence of the moderator variables within this subset of studies was not conducted. This is because with only six total effect sizes, the number of effect sizes at each level of any moderator variable would be so small (e.g., even with only two levels, at least one level would have three or fewer effect sizes) as to make the results meaningless. Therefore, these effect sizes were dropped from subsequent statistical analyses.

Examination of the means within each of the remaining exercise paradigms showed that significantly larger effect sizes were found with cross-sectional/correlational designs ($M = 0.53$, $SD = 0.77$, $ES\ n = 117$, $p < .05$) than were found for chronic designs ($M = 0.33$, $SD = 0.58$, $ES\ n = 358$, $p < .05$) or acute designs ($M = 0.16$, $SD = 0.60$, $ES\ n = 371$, $p < .05$). The studies using chronic paradigms showed significantly larger effect sizes than those using acute paradigms. This supports Hypothesis 1, which suggested that studies using cross-sectional comparisons or chronic exercise programs would show the largest effect sizes. Physiological mechanisms were

not actually assessed in the examination of this hypothesis; therefore, this finding provides only indirect support for physiological mechanisms for enhanced cognition because it suggests that longer commitments to exercise may be necessary for the biggest gains in cognitive performance. However, it is important to note that all of the effect sizes were significantly different from zero, thus suggesting that there is a significant effect of exercise on cognition regardless of the exercise paradigm that is used. From this point on, all analyses were conducted within the subsets of the effect sizes created by the exercise paradigm variable.

Acute Exercise

Moderator Variables. Examination of the moderator variables within studies using this exercise paradigm showed that the variables that significantly affected the effect sizes were: the method of sampling, the number of threats to internal validity, the cognitive test category, the size of the exercise group, and the sex of the participants (for F values, means, standard deviations, and sample sizes, see Table 1).

Examination of the means related to method of sampling indicated that the largest effect sizes were found when random sampling was used ($M = 0.65$); however, this method was only used in seven of the effect sizes. A post hoc analysis for sampling method indicated that effect sizes were significantly larger when intact groups ($M = 0.50$) were used than when volunteers ($M = 0.13$), unreported ($M = 0.13$), or "other" ($M = -0.08$) methods of sampling were used.

Post hoc examination of the mean effect sizes relative to the number of threats to internal validity showed that none of the categories with five or more effect sizes was significantly different from the others. However, despite the lack of a significant difference between the levels of the factor, it is interesting to note that as the number of threats increased, the mean effect tended to increase. Thus, the mean effect size increased as follows: zero threats ($M = -0.04$), one threat ($M = 0.13$), three threats ($M = 0.21$), two threats ($M = 0.23$), and four threats ($M = 1.76$). This suggests, then, that as experimental rigor decreased, effect size increased.

The cognitive test used also influenced the effect sizes. However, the post hoc analysis indicated that none of the levels were significantly different from each other.

The number of participants in the exercise group also had an impact on the results. When the exercise group was larger than 20 people, the effect sizes were significantly larger ($M = 0.61$) than when the group was an unreported size ($M = 0.07$) or when the participants exercised alone ($M = 0.09$). An examination of the mean effect sizes showed that the benefits of exercise upon cognitive functioning increased as the size of the exercise group increased. That is, the means were as follows relative to the group size: alone ($M = 0.09$), 10 or less ($M = 0.16$), between 11 and 20 people ($M = 0.61$), and more than 20 people ($M = 0.61$).

The sex of the participants tested also influenced the effect sizes. Samples of men and women ($M = 0.70$) or samples in which participants' sex was unreported ($M = 0.57$) showed significantly larger effect sizes than samples with all women ($M = 0.14$) or all men ($M = 0.03$).

Table 1. Analyses for the moderator variables

Moderator variable	<i>F</i> test	<i>p</i>	Effect size	<i>SD</i>	<i>n</i>	<i>p</i>
Overall			0.25	0.69	1,260	*
Independent effects			0.30	0.78	873	*
Outliers removed			0.29	0.63	852	*
Exercise paradigm	$F(3, 848) = 12.43$.001				
Acute			0.16 ^c	0.60	371	*
Chronic			0.33 ^b	0.58	358	*
Cross-sectional/correlational			0.53 ^a	0.77	117	*
Mixed			0.54	0.43	6	*
<i>Acute exercise studies</i>						
Sampling method	$F(5, 365) = 6.44$.0001				
Other			-0.08 ^b	0.39	58	
Volunteer			0.13 ^b	0.43	99	*
Not reported			0.13 ^b	0.70	157	*
Random stratified			0.47	0.31	2	*
Intact group			0.50 ^a	0.68	48	*
Random sampling			0.65	0.22	7	*
Threats to internal validity	$F(4, 366) = 9.52$	<.0001				
0			-0.04 ^b	0.33	45	
1			0.13 ^b	0.53	191	*
3			0.21 ^b	0.72	62	*
2			0.23 ^b	0.68	69	*
4			1.76 ^a	0.47	4	*
Cognitive test category	$F(10, 360) = 5.23$	<.0001				
Reasoning			-0.06	0.83	2	
Verbal			-0.02 ^b	0.27	22	
Reaction time			0.06 ^b	0.49	116	
Memory			0.10 ^b	0.63	116	
Other			0.18	0.30	4	
Math			0.21	0.62	40	*
Perception			0.29	0.64	57	*
Not reported			0.63	0.30	5	*
Mixed bag of tests			1.20	—	1	
Academic achievement			1.23	0.44	4	*
Motor skills			1.47 ^a	1.03	4	*
Exercise group size	$F(5, 365) = 4.96$	<.0002				
Not reported			0.07 ^b	0.31	35	
Alone			0.09 ^b	0.60	250	*
Group (≤10 people)			0.16	0.81	17	
Group (unknown number of people)			0.31	0.60	38	*
Group (11–20 people)			0.61	0.32	5	*
Group (>20 people)			0.61 ^a	0.65	26	*
Sex	$F(3, 367) = 25.09$	<.0001				
Male			0.03 ^b	0.46	269	
Female			0.14 ^b	0.49	30	
Not reported			0.57 ^a	0.99	23	*
Mixed			0.70 ^a	0.75	49	*

(continued)

Table 1 (continued)

Moderator variable	F test	p	Effect size	SD	n	p
<i>Chronic exercise studies</i>						
Threats to internal validity	$F(6, 351) = 8.22$	<.0001				
0			0.06 ^b	0.39	28	
2			0.14 ^b	0.36	137	*
6			0.35	0.02	2	*
3			0.37	0.37	24	*
1			0.45	0.48	55	*
4			0.57 ^a	0.91	75	*
5			0.57 ^a	0.39	37	*
Subject Assignment	$F(5, 352) = 15.29$	<.0001				
Random			0.15 ^b	0.42	171	*
Not reported			0.26 ^b	0.40	20	*
Intact			0.35 ^b	0.62	74	*
Matching			0.38 ^b	0.41	22	*
Random stratified			0.49	0.38	22	*
Single group			0.88 ^a	0.79	49	*
Fitness measurement	$F(5, 352) = 9.54$	<.0001				
IPS or other			-0.05		1	
Max test			0.17 ^{b,2}	0.37	170	*
Not reported			0.29 ^b	0.47	59	*
Submaximal test			0.45 ¹	0.70	59	*
Self-report			0.50	0.24	4	*
Not measured			0.69 ^a	0.78	65	*
Age group	$F(7, 350) = 7.60$	<.0001				
Not reported			-0.19 ^b	0.86	5	
Adult (30–45 yr)			0.06 ^b	0.35	10	
Oldest adult (60–90 yr)			0.19 ^{b,2}	0.37	163	*
Elementary (6–13 yr)			0.36	0.33	39	*
More than one age range			0.40	0.70	90	*
College (18–30 yrs)			0.64 ¹	0.69	32	*
High school (14–17 yr)			0.77	0.40	8	*
Older adult (45–60 yr)			1.02 ^a	1.15	11	*
Exercise environment	$F(6, 351) = 10.42$	<.0001				
Fitness center			0.06 ^{b,2}	0.35	62	
Other			0.10 ^{b,2}	0.32	60	*
Hospital			0.25 ^b	0.45	35	*
Home (or on own)			0.28	0.33	31	*
Laboratory			0.43 ¹	0.64	80	*
Not Reported			0.45	0.38	15	*
Class setting			0.67 ^a	0.75	75	*
Exercise leader	$F(4, 353) = 8.93$	<.0001				
Experimenter			-0.04	0.09	4	
Fitness trainer			0.07 ^b	0.28	66	*
No leader			0.31 ^b	0.53	183	*
Not reported			0.34	0.45	22	*
Teacher			0.60 ^a	0.76	83	*

(continued)

Table 1 (continued)

Moderator variable	F test	p	Effect size	SD	n	p
Exercise group size	$F(5, 352) = 18.39$	<.0001				
Group (>20 people)			0.12 ^b	0.34	85	*
Group (unknown number of people)			0.25 ^b	0.45	89	*
Alone			0.34 ^b	0.56	135	*
Not reported			0.45 ^b	0.38	15	*
Group (11–20 people)			0.47 ^b	0.44	9	*
Group (≤10 people)			1.22 ^a	0.94	25	*
<i>Cross-sectional/correlational studies</i>						
Equivalency of the comparison groups	$F(2, 114) = 6.77$	<.0017				
Yes			0.11 ^b	0.59	22	
Not reported			0.18	0.37	11	
No			0.69 ^a	0.79	84	*
Comparison group	$F(1, 115) = 11.41$	<.001				
Correlation			0.19 ^b	0.47	29	*
Nonathletes			0.71 ^a	0.77	84	*
Cognitive test category	$F(10, 106) = 3.08$	<.0018				
Math			−0.53 ^b	0.41	5	*
Mental age tests			0.00	0.22	5	
Perception			0.17	—	1	
Verbal			0.17	0.33	8	
Academic achievement			0.27	0.46	20	*
Memory			0.45	0.35	4	*
Other			0.48	0.97	6	
Reaction time			0.74 ^a	0.82	60	*
Reasoning			0.83	0.16	4	*
IQ			1.24	1.13	3	
Not reported			1.69	—	1	
Exercise group size	$F(5, 111) = 5.13$	<.0003				
Group (11–20 people)			−0.49	0.71	4	
Group (>20 people)			−0.24	0.24	5	*
Not reported			0.24	0.66	24	
Group (unknown number of people)			0.60	0.72	41	*
Alone			0.80	0.80	32	*
Group (≤10 people)			0.85	0.50	11	*
Exercise intensity	$F(3, 113) = 7.12$	<.0001				
Low			−1.12 ^b	0.62	2	*
High			−0.70 ^b	0.16	4	*
Not reported			0.58 ^a	0.72	103	*
Moderate			0.84 ^a	0.49	8	*

Note. Superscripts that are different represent effects which are significantly different from one another as determined by Scheffé post hoc tests. Asterisks indicate that the effect is significantly different from zero.

A Priori Hypotheses. Within the studies using acute exercise, the moderator variables of age group and mental ability were examined. Neither had a significant impact on the results: $F(2, 368) = 4.058, p > .01$, and $F(5, 365) = 2.174, p > .05$, respectively. Therefore, neither Hypothesis 2 nor Hypothesis 3 was supported.

To test Hypothesis 4, the effect sizes for measures of RT were examined relative to the intensity level of the exercise. To do this, RT was explicitly coded as being simple RT or choice or discriminant RT, and the size of the effects were examined as a function of these levels. The results showed that regardless of exercise intensity, larger effect sizes were found for measures of simple RT than for measures of choice or discriminant RT (low intensity: simple RT $M = 0.16$, choice RT $M = -0.22$; moderate intensity: simple RT $M = 0.21$, choice RT $M = -0.56$; high intensity: simple RT $M = 0.08$, choice RT $M = -0.30$). Thus, Hypothesis 4 was not supported. The lack of support for this hypothesis seems counter to the logic guiding research in this area. That is, because choice and discriminant RT have greater cognitive components (because more decision making is required) than does simple RT, it was expected that the greatest benefit of fitness would be seen on the choice and discriminant RT tasks. However, the results are in the opposite direction.

Hypothesis 5 stated that for acute exercise, there would be an inverted-U relationship between the duration of the exercise bout and the effect on cognitive performance. This hypothesis was not supported because the linear relationship between exercise duration and cognitive performance was not significant, $F(1, 369) = 0.18, p > .05, R^2 = .02$, and a curvilinear fit did not produce a change in R^2 .

Hypothesis 6 predicted that studies with acute exercise of short duration (2-15 min) and moderate intensity would produce larger effect sizes than would studies with exercise of longer duration (> 15 min) or with light or difficult exercise intensity. To test this hypothesis, acute exercise studies were coded based upon these distinctions. The ANOVA was not significant, $F(3, 327) = 2.87, p > .03$; therefore, Hypothesis 6 was not supported.

Chronic Exercise

Examination of the moderator variables for studies within this category indicated that the following variables had a significant impact on the effect sizes: number of threats to internal validity, the method of assigning participants to groups, the fitness measure used, the exercise environment, the exercise leader, and the size of the exercise group, (for F values, means, standard deviations, and sample sizes; see Table 1).

Effect sizes resulting from studies that had four threats ($M = 0.57$) or five threats ($M = 0.57$) to internal validity were significantly larger than were effect sizes resulting from studies that had no threats ($M = 0.06$) or two threats ($M = 0.14$). In general, this finding again supports a conclusion that as experimental rigor decreases, the magnitude of the effect increases. The method of assignment of the participants to treatment conditions also had a significant impact on the effect sizes. When participants were assigned in a single group ($M = 0.88$), the effect sizes were not significantly different from when a random stratified method ($M = 0.49$) was used; however,

single group assignment resulted in significantly larger effect sizes than any other method of participant assignment (range: $M = 0.15$ to $M = 0.38$).

Effect sizes resulting from studies in which fitness was not measured ($M = 0.69$) or from studies that used submaximal measures of fitness ($M = 0.47$) were significantly larger than were effect sizes from studies that used maximal tests of fitness ($M = 0.17$). Additionally, effect sizes from studies in which fitness was not measured were significantly larger than effects from studies in which fitness was measured, but in which the particular measure of fitness was not reported ($M = 0.29$). Again, this suggests that as experimental rigor increased, effect size decreased.

The environment in which the exercise was conducted also affected the results such that studies in which the exercise was performed in a classroom setting ($M = 0.67$) showed significantly larger effect sizes than did studies in which the exercise was performed in a hospital ($M = 0.25$), in some other setting ($M = 0.10$), or in a fitness center ($M = 0.06$). Additionally, significantly larger effect sizes were found for studies in which the exercise was completed in a laboratory ($M = 0.43$) rather than a fitness center or some other setting. The leader of the exercise also affected the results, such that effect sizes from studies in which a teacher led the exercise ($M = 0.60$) were significantly larger than effect sizes from studies in which there was no leader ($M = 0.31$) or in which the exercise was led by a fitness trainer ($M = 0.08$). These findings with regard to exercise environment and to the exercise leader suggest two possible interpretations. It is possible that when the exercise participants have a sense that the exercise is required (as in a classroom setting or in a laboratory) and when the exercise is led by an authority figure (as in the teacher), the cognitive benefits of exercise are higher. However, it is equally possible that in these same situations the exercise leader was not blind to the purpose of the study and this knowledge may have influenced the outcome of the results of these studies.

The size of the exercise group also had an impact on the effect sizes: Effect sizes from studies in which the group size was 10 or fewer ($M = 1.22$) were significantly larger than effect sizes from studies in which the exercise group was of any other size ($M = 0.12$ - 0.47).

A Priori Hypotheses. Within the studies using chronic exercise, the moderator variables of age group and mental ability were examined. The mental ability of the participants did not have a significant impact on effect size, $F(2, 355) = 2.51, p > .05$; therefore, Hypothesis 2 is not supported. Age group of the participants did have a significant impact on effect size, $F(7, 350) = 7.60, p < .001$. The nature of this effect was such that significantly larger effect sizes were found for studies in which the participants were 46-60 years old ($M = 1.02$) than for studies in which the participants were of unreported ages ($M = -0.19$), 31-45 years old ($M = 0.06$), or 61-90 years old ($M = 0.19$). Additionally, significantly larger effect sizes were found for studies in which the participants were 18-30 years old ($M = 0.64$) than for studies in which the participants were 61-90 years old. This finding fails to support Hypothesis 3 because the order of the effect sizes from largest to smallest was as follows: older (46-60 years), high school (14-17 years), college (18-30 years), elementary (6-13 years), oldest (61-90 years), adult (31-45 years). However, it does suggest a slightly modified hypothesis: Exercise may be beneficial to participants who are slightly resource dependent (high school, older), but may not benefit those who are more resource dependent (elementary, oldest) or who are not at all resource dependent (college, adult).

Hypothesis 7 was examined by assessing the relationship between effect size and the duration of each training session, the days of training per week, and the total number of weeks of training. None of these correlations was significant, $r_s = -.12$ to $.05$, $p_s > .01$. Additionally, effect size was examined as a function of whether the authors reported evidence of a training effect, and the test was not significant, $F(2, 355) = 3.44$, $p > .03$. Thus, no support was found for Hypothesis 7.

Clinical Trials. Within this exercise paradigm, clinical trial studies (those that use random assignment of participants to groups and that use a chronic exercise intervention) may be of special interest. These studies ($n = 17$) were identified, and the overall mean effect size for these studies was 0.18 ($SD = 0.42$, $ES\ n = 188$, $p < .05$), with an overall sample size of approximately 420 participants. This suggests, then, that in well-controlled studies using chronic paradigms, the mean effect size is positive, but small. Interestingly, however, within this subsample of studies, there was no relationship between effect size and duration of each training session, days of training, weeks of training, or evidence of a training effect. Thus, the length of the training period was not associated with the size of the effect regardless of how the length of the training period was quantified. Hypothesis 7 again was not supported.

Cross-Sectional/Correlational Designs

The moderator variables that had significant impacts upon the effect sizes were the equivalency of the comparison groups, the comparison group used, the cognitive test category, the size of the exercise group, and the intensity of the exercise (for F value, means, standard deviations, and sample sizes; see Table 1).

Examination of the means for the equivalency of the comparison groups showed that when the comparison groups were unequal ($M = 0.69$) the effect sizes were significantly greater than when the comparison groups were equal ($M = 0.11$). This finding illustrates the difficulty in gleaning information about the relationship between exercise and cognitive functioning using a cross-sectional design because the effect sizes are so much larger with groups that were not equal in terms of relevant variables influencing the effect. The particular comparison group used also had a significant impact on the results, such that studies using correlations of fitness to cognitive ability had significantly smaller effect sizes ($M = 0.19$) than studies using comparisons between athletes and nonathletes ($M = 0.71$).

The cognitive test category had a significant impact upon the size of the effects such that the effect sizes from RT measures ($M = 0.74$) were significantly larger than the effect sizes from math test performance ($M = -0.53$).

The size of the exercise group also had a significant influence upon the size of the effects. To increase the cell size, the effect sizes from studies using 11-20 people and those using more than 20 people in the exercise group were combined. Following this, the overall F test was still significant, $F(4, 112) = 6.39$, $p < .0001$. Post hoc analyses indicated that individuals who exercise in groups of 10 or fewer ($M = 0.85$), who exercised alone ($M = 0.80$), or who exercised in a group of an unreported size ($M = 0.24$) experienced significantly larger effect sizes than did those who exercised in groups of more than 10 ($M = -0.35$). Additionally, the intensity of the exercise had a significant influence on the results. However, the interpretation of these results is

not meaningful because the intensity of the exercise was not quantified in the majority of the studies (103 of 117).

A Priori Hypotheses. Neither the mental ability of the participants, $F(1, 115) = 0.26, p > .05$, nor the age group of the participants, $F(6, 110) = 1.63, p > .05$, had a significant impact on the results. Therefore, neither Hypothesis 2 nor Hypothesis 3 was supported.

To examine Hypothesis 8, simple cognitive tasks were compared to more complex cognitive tasks. To do this, RT measures were coded as being either simple (RT) or complex (choice, discriminant RT). Results showed that the effect sizes were larger for the simple measures than for the complex measures, thus failing to support Hypothesis 8. In an attempt to examine the other cognitive categories, effect sizes were also coded as simple (perception) or complex (math, verbal, reasoning, IQ, academic achievement, mental age), but the hypothesis could not be examined because only one effect size was available from a simple task.

To examine Hypothesis 9, effect sizes that reflected academic achievement were examined relative to these two exercise paradigms. It was found that small positive effect sizes existed for both cross-sectional comparisons ($M = 0.22$) and correlational comparisons ($M = 0.31$), thus supporting Hypothesis 9.

Third-Order Causation

Moderator variables that were found to have a significant impact upon effect size were examined to identify potential cases of third-order causation. Only those variables for which the loadings of the levels of the two variables were of concern are discussed.

Within the acute subset, examination of the loading of the effect sizes within the cognitive test categories showed that the effect sizes in which a verbal test was used to measure cognition were almost all (21 of 22) from studies using male participants only and were also almost all (20 of 22) from studies in which the exercise was performed alone. Since the overall effect sizes for studies using males ($M = 0.03$) and for studies in which the exercise was performed alone ($M = 0.09$) were so small, this suggests that the low effect sizes for the verbal tests ($M = -0.02$) may be a result of the sex of the participants or the exercise group size rather than the nature of the cognitive measure.

Within the chronic studies, the loading of the effect sizes for two of the variables with exercise environment showed that the exercise environment may have affected the findings for other variables. With regard to exercise environment and exercise group size, 24 of the 25 effect sizes for exercise groups that consisted of fewer than 10 people ($M = 1.22$) were derived when the exercise was conducted in a classroom setting. This suggests that the large effect sizes for exercise groups of fewer than 10 may actually be due to the exercise setting rather than to the group size. With regards to exercise environment and exercise leader, 50 of 66 effect sizes for fitness trainer ($M = 0.07$) were effect sizes that were found in a fitness center ($M = 0.06$), and all of the effect sizes for exercise led by a teacher ($M = 0.60$) were from studies in which the exercise was conducted in a classroom. Thus, the large effect sizes for the exercise being led by a

teacher may be due the classroom setting and the small effect sizes for the exercise being led by a fitness trainer may be due to the exercise being conducted in a fitness center.

Additionally, within this subset of studies, all of the effect sizes from studies with older adults ($M = 1.02$) were from studies in which fitness was not assessed in any way ($M = 0.69$). Thus, the large effect sizes for older adults may be due to the failure to use any measure of fitness. Finally, levels of exercise group size matched up with levels of age group such that two thirds of the effect sizes (66 of 94) for exercise groups which had 11 or more people were also effect sizes which were found with the oldest adults (age 60-90 years). Therefore, the small effect sizes for the large exercise groups ($M = 0.15$) may have been more a result of the age of the participants ($M = 0.19$) than the exercise group size.

Within the correlational and cross-sectional studies, the only loading of interest was between the cognitive test category and the comparison group. Most of the effect sizes when the comparison was between athletes and nonathletes (58 of 84) ($M = 0.70$) were from studies using RT measures ($M = 0.74$) and nearly all of these RT measures (58 of 60) were from this comparison group. Thus, the large effect sizes for the particular levels of cognitive test category and comparison group cannot necessarily be attributed to either factor alone.

Outliers

The effect sizes that were identified as outliers were examined individually and as a group based upon their valence to determine whether there was something unique about these results that might contribute to their magnitude. The negative effect sizes ($n = 8$) identified as outliers all came from studies in which the effect was calculated by making a comparison to the participants' own pretests, and in which the participants were all men with normal mental capabilities; in addition, five of these effect sizes came from studies conducted in a laboratory. The positive effect sizes ($n = 13$) identified as outliers all came from published studies in which participants had normal mental capabilities. Additionally, 10 of the effect sizes came from studies using within-subjects designs, 9 of the studies had high internal validity, and 4 had moderate internal validity.

Discussion

The results of this meta-analysis show that when the findings of 134 studies are combined mathematically, exercise and fitness have a small (Cohen, 1992) positive effect on cognitive performance ($ES = 0.25$). In particular, when only one comparison group is used per study and when outliers are omitted, the overall effect is slightly greater than 0.25 standard deviations ($ES = 0.29$).

Within this group of studies, different exercise paradigms were used such that some studies examined the effects of an acute bout of exercise on cognition, others examined the effects of a chronic exercise training program on cognition, and still others used a cross-sectional comparison or a correlational analysis to look at cognition as a function of fitness level. The results showed that the effect sizes were largest when a cross-sectional or correlational method

was used ($ES = 0.53$), next largest for a chronic training program ($ES = 0.33$), and smallest for an acute bout of exercise ($ES = 0.16$).

Taken at face value, these results could indicate that the influence of exercise on cognition is inconsequential when small and temporary changes in physiological parameters occur as a result of acute exercise, but that the influence becomes larger as either the size or the permanence of these changes increases. In other words, exercise may not have a meaningful impact on cognition when it is administered in acute bouts, but exercise that is administered as a chronic treatment to produce fitness gains, or exercise that has been adopted by an individual for a sufficiently long period of time to produce fitness gains, may be a useful intervention for enhancing cognitive abilities. This conclusion, then, would support the physiological mechanisms as explanations for the beneficial effects of exercise or fitness upon cognition. Additionally, this conclusion would suggest that the adoption of a chronic exercise program may be a useful intervention for enhancing cognitive functioning.

However, these findings should not be taken at face value because there are a number of serious concerns that make these results substantially less compelling. One such concern is that conclusions from the cross-sectional and correlational studies are limited by the flaw of quasi-experimental designs. That is, it is possible that the differences in cognition as a function of fitness are not differences that are actually caused by the exercise, but instead are preexercise cognitive differences associated with people who choose to adopt a lifelong commitment to exercise. Additional strength is added to this alternative explanation by the fact that people who exercise tend to have higher levels of education and to be of higher socioeconomic status than people who do not choose to exercise. Thus, the conclusion that can be drawn from this result with the cross-sectional and correlational studies is limited to saying that there appears to be a relationship between fitness and cognition.

The typical response to this limitation of the cross-sectional and correlational studies is to instead focus the examination on studies that used exercise as an intervention in a previously sedentary population so that a cause-and-effect relationship could be established. Examination of the effect sizes for the chronic exercise studies then suggests that exercise does have a small positive impact upon cognitive functioning. However, the second requirement for establishing cause and effect is that participants in the study be randomly assigned to either the treatment or control condition. When the chronic exercise studies are examined in terms of this criterion, there is further cause for hesitation. Within the chronic studies, the largest effect sizes were found when participants were assigned to treatment conditions as a group or when participants were allowed to self-select their treatment condition. Again, this limitation means that a cause-and-effect relationship has not been established because there may be preexisting differences among the treatment groups that explain the subsequent differences in cognitive ability.

To truly establish a cause-and-effect relationship for exercise and cognition, one must use a chronic exercise program in which sedentary participants are randomly assigned to treatment conditions. To examine this relationship, those studies that used true-experimental designs in randomized trials were examined separately. The results showed that the overall effect size was small ($ES = 0.18$) but still positive and significantly different from zero. This would suggest then that implementing a chronic exercise program in sedentary individuals can cause increased

cognitive functioning. However, even this conclusion is limited. Most theories explaining the potential beneficial effects of exercise on cognition suggest that physiological changes which result from exercise are the mechanisms underlying this relationship. Although it was not possible to examine these mechanisms directly, it was possible to examine them indirectly by looking at the influence of some of the moderator variables on effect size. The findings with regard to the length and effectiveness of the chronic exercise programs provide evidence against the physiological mechanisms. In particular, there was no relationship between effect size and any of the variables representing the length of the exercise program. That is, neither the number of weeks of exercise nor the number of days of exercise per week were related to effect size.

Additionally, any evidence of a training effect was not related to effect size. Because most physiological mechanisms are thought to be evoked through increases in fitness, the inability of these variables to affect the results provides evidence against the physiological mechanisms as explanations for the beneficial effects of exercise. Thus, although small and meaningful changes in cognition may be obtainable through the implementation of a chronic exercise program, the results from this analysis suggest that the underlying explanation for these changes must be one of the following: (a) physiological mechanisms independent of aerobic fitness; (b) physiological mechanisms related to aerobic fitness, but occurring prior to changes in aerobic fitness; or (c) psychological mechanisms independent of aerobic fitness and exercise.

At this point, the exercise proponent seeking a thread of evidence supporting the hypothesis that exercise positively affects cognitive abilities may feel discouraged. However, it should be remembered that a mathematical summary of 134 studies suggests that both acute exercise and chronic training programs benefit cognitive performance. Additionally, further examination of the effects indicated that chronic exercise programs with randomized trials resulted in small positive gains in cognitive performance. However, even the most avid exercise proponent must recognize that the weakness of the design of these studies has resulted in serious limitations with any conclusions regarding the relationship between exercise and cognition and have also resulted in ambiguity in terms of the nature of the relationship between exercise or fitness and cognition. We hope that the findings from this meta-analysis regarding both the impact of the moderator variables on effect size and the need for well-designed research in this area will help guide future research. In particular, if exercise does have a beneficial effect on cognition, this effect will only be established through the completion of true-experimental studies designed to specifically examine the potential underlying mechanisms.

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² References marked with an asterisk indicate studies included in the meta-analysis.

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